and should be removed; the superficial ulcer (based on the subsequent pathological report) may be contributing little to the submucosal haemorrhage. These haemorrhages suggested a widespread pathological process in the stomach wall of unknown aetiology. Additional investigations were clearly required. The initial step in these further procedures was the aspiration of blood from the gastric veins and arteries at routine gastric operations. The laboratory testing of these specimens of blood was carried out by my colleagues at Withington Hospital and the first of our reports appeared in 1967.¹

H T Cox

Wilmslow, Cheshire

¹ Cox, H T, Poller, L, and Thomson, J M, *Lancet*, 1967, **1**, 1300.

"Squelch sign" in detection of small herniae

SIR,—It may sometimes be difficult to detect the presence of a small inguinal rupture, particularly if it is hard to be convinced of the presence of a cough impulse. The following method has been found to be valuable.

The patient is examined standing. If the clinician is right-handed he examines the patient while standing on his right. He places his hand flat over the patient's inguinal canal and applies pressure with a quick, jerky movement of the fingers (similar to that used in "dipping" to detect the presence of ascitic fluid). If a hernia is present the contents of the sac are felt sliding back into the abdominal cavity, producing a "squelching" sensation.

A similar sensation can sometimes be felt with a large lipoma of the cord. Nevertheless, when one is in any doubt about the presence of a hernia and a cough impulse cannot be elicited with certainty a positive "squelch sign" may be a useful aid to diagnosis.

W G EVERETT

Addenbrooke's Hospital,
Cambridge

Papilloedema in patients taking perhexiline maleate

SIR,—We were interested in the report of "Raised intracranial pressure due to perhexiline maleate" by Dr W P Stephens and others (7 January, p 21), but we question the conclusion reached in their title. The three patients described had bilateral papilloedema; investigation excluded an intracranial mass or hydrocephalus and the fundal appearances improved when perhexiline was stopped. Measurement of cerebrospinal fluid (CSF) pressure was not made during the initial evaluation, but one man had a pressure of "130 mm Hg" (presumably 130 mm H₂O) six weeks after the perhexiline was stopped, when his fundi had returned to normal.

Papilloedema has been noted previously in three patients who presented with a peripheral neuropathy caused by perhexiline.¹ The CSF pressures were not included in this report, but a high CSF protein concentration was noted, up to 10 g/l. Although there was no clinical evidence for a neuropathy among Dr Stephens's patients, nerve conduction studies might have been revealing as one patient (case 3) complained of numbness and weakness of the limbs and all had ataxia of gait.

We recently investigated a 52-year-old man with a peripheral neuropathy and papilloedema; he had taken perhexiline maleate for one year. No structural intracranial lesion was defined and his CSF pressure was normal. The CSF protein level was elevated at 1.46 g/l. Four months after stopping perhexiline there had been substantial improvement in his neurological state and fundal appearances.

We feel that the cause of papilloedema in patients taking perhexiline needs to be identified for there is as yet no proof of elevated intracranial pressure. (We accept that intermittent pressure elevation might explain the findings in our patient.)

Papilloedema is sometimes seen in association with the Guillain-Barré syndrome. Abnormalities in CSF protein and changes in CSF pressure have been implicated in this context, but the mechanism for papilloedema remains unknown.^{2 3}

W M HUTCHINSON
JOHN WILLIAMS
JEFFREY GAWLERDepartment of Neurological
Sciences,
St Bartholomew's Hospital,
London EC1

¹ Bousser, M G, *et al*, *Coeur et Médecine Interne*, 1976, **15**, 181.

² Drew, A L, and Magee, K R, *Archives of Neurology and Psychiatry*, 1951, **66**, 744.

³ Morley, J B, and Reynolds, E H, *Brain*, 1966, **89**, 205.

Thyroid disease and asthma

SIR,—Your leading article (5 November, p 1173) omits discussion of the possibility that the link between these two types of disease might be the iodine contained in the medicines given to asthmatics.

The syndrome of iodine-induced thyrotoxicosis (Jod-Basedow) is well recognised. A recent review by Weaver *et al*¹ suggests that the increase in a variety of thyroidal abnormalities may be related to the iodine supplements added to our diet. Those who take large and continuing doses of potassium iodide, among them many asthmatics, are the more likely to show evidence of toxicity from this potentially harmful element.

ELINOR D U POWELL

Victoria, BC,
Canada

¹ Weaver, D K, Nishiyama, R H, and Batsakis, J G, *Annals of Clinical and Laboratory Science*, 1976, **6**, 545.

Pathogenesis of acute appendicitis

SIR,—I would like to endorse the findings of Mr L W L Horton (24-31 December, p 1672) with regard to the pathogenesis of acute appendicitis. At the suggestion of Mr Denis Burkitt we also have studied the histological findings in 30 consecutive cases of acute appendicitis. Appendices were opened longitudinally when fresh, as described by Mr Horton. In our series the inflammation was total or confined to the distal part of the appendix in 90% of cases. Inflammation of the proximal part of the appendix occurred in only one case.

Similarly, in our series, faecoliths were found in only 10% of cases, but an obstructed lumen due to faeculent or purulent material was found in 60%. These findings would tend

to confirm the low-fibre theory of obstruction causing acute appendicitis suggested by Mr Horton.

J R JOHNSON

Royal Berkshire Hospital,
Reading, Berks

Vasovagal shock after insertion of intrauterine device

SIR,—With reference to your expert's answer to a question on this subject (24-31 December, p 1645), while it is to be hoped that all clinicians will use care and gentleness when dealing with patients and while this approach would no doubt reduce patients' anxiety, it remains a fact that inhibition of cardiac action as a result of pelvic examination can at times be dangerous. This matter was dealt with by you in 1970 in a leading article¹ which was followed by a letter from me² pointing out the increased risk during the insertion of intrauterine devices.

To attempt to remove a device while a patient is suffering from vasovagal shock could result in more serious complications. The correct treatment is to inject atropine intravenously. (Unless premedication has included the use of atropine even light anaesthesia will not prevent this potentially fatal complication.) The value of the atropine administration can be confirmed by observing that the pulse rises to a normal value some 60 s after the injection. The answer to the question put to your expert should have been: "A syringe and solution of atropine suitable for intravenous administration." No clinic at which intrauterine contraceptives are inserted ought to be without this drug, and other resuscitation equipment should be available. As your expert writes, "Heaven forbid that any other resuscitative measures should need to be considered," but this cannot mean that the possibility is to be ignored.

D N MENZIES

Liverpool Maternity Hospital,
Liverpool

¹ *British Medical Journal*, 1970, **3**, 61.

² Menzies, D N, *British Medical Journal*, 1970, **3**, 221.

* * * Our expert replies: "What Mr Menzies suggests is sensible and reasonable, though what I recommended should really be enough. His suggestion that removal of the device might increase the shock is probably more theoretical than real."—ED, *BMJ*.

A case of self-diagnosis

SIR,—I read Dr Eoin O'Brien's article (24-31 December, p 1648) with enjoyment, but I suspect that he is mistaken in attributing his malaise to the Portuguese man-o-war *Physalia physalis*. Strong winds blow physalia from warmer waters to the south and west coasts of the British Isles by acting on its sail-like float. Although occasionally it arrives in great numbers,^{1 2} it makes little headway into the Irish Sea, where it is rare.³ I know of no records from the Irish shore as far north as the Martello Tower from which Buck Mulligan gazed out over this "snotgreen scrotum-tightening" sea.⁴ No unusual influx of physalia was reported in the British Isles in 1977 when, I presume, Dr O'Brien had his unpleasant experience. It is unlikely, therefore, that this species was present at the Forty Foot.